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CONTRASTING BEHAVIORAL EFFECTS OF ACUTE NICOTINE AND CHRONIC SMOKING IN DETOXIFIED ALCOHOLICS

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Abstract

Background—Current literature suggests that acute nicotine administration provides a compensatory mechanism by which alcoholics might alleviate attentional deficits. In contrast, chronic smoking is increasingly recognized as negatively affecting neurobehavioral integrity. These opposing effects have not been simultaneously examined. Thus, we sought to a) extend previous work by exploring the effects of acute nicotine effects on vigilance components of attention and replicate previous findings suggesting that treatment-seeking alcoholics experience benefit to a greater extent than do other groups; and b) to examine the impact of chronic smoking on these tasks and across subgroups.

Methods—Substance abusing participants (N=86) were recruited and subgrouped on the basis of dependency criteria as either alcoholics, alcoholics with co-morbid stimulant dependence, or stimulant dependent individuals. Groups of cigarette-smoking (N=17) and non-smoking (N=22) community controls were recruited as comparison groups. Smoking subjects were assigned a placebo, low, or high dose nicotine patch in a double-blind placebo controlled fashion. Non-smoking controls were administered either a placebo or low dose. Testing occurred after dose stabilization.

Results—General linear models indicated greater sensitivity to acute nicotine administration among alcoholics than other groups when controlling for the effect of intensity of smoking history, as reflected by pack-years. Pack-years correlated negatively with performance measures in alcoholics but not stimulant abusing subgroups or smoking controls. Finally, regression analyses demonstrated that pack-years predicted poorer performance only for the alcoholic subgroup.

Conclusions—These results support previous work finding a compensatory effect of acute nicotine administration on attentional performance in alcoholics and reinforce the consideration of recent nicotine use as a confound in neurocognitive studies of alcoholics. Of particular interest is the finding that smoking history as reflected in pack-years predicted poorer performance, but only

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among alcoholics. Further systematic study of these opposing effects among alcoholics and other groups using a broader array of tasks is needed.

Keywords

alcohol; neurocognition; attention; nicotine; smoking

1. Introduction¹

The co-morbidity of alcohol and nicotine dependence is a significant public health concern (Littleton et al., 2007). Research suggests chronic heavy alcohol consumption is associated with neurocognitive compromise (Oscar-Berman and Marinovic, 2007) and structural brain changes (Pfefferbaum et al., 1995; Pfefferbaum et al., 2006). Most of these studies have not systematically accounted for either acute nicotine exposure or smoking history, yet over 80% of treatment-seeking alcoholics are current smokers (Littleton et al., 2007).

Although subject to some debate (Bell et al, 1999; Heishman et al., 2010), acute nicotine administration appears to enhance neurocognition. Beneficial effects do not appear limited to the amelioration of nicotine withdrawal symptoms; having been observed in non-smokers and clinical populations (Wesnes and Warburton, 1983; Rezvani and Levin, 2001; Lawrence et al., 2002; Kumari et al., 2003; Levin et al., 2006).

It is hypothesized that nicotine use may partially compensate for alcohol-related deficits (Nixon et al., 2007). Consistent with this hypothesis, alcoholics demonstrated greater benefit from nicotine administration than controls and other substance abusing subgroups on specific neuropsychological (Nixon et al., 2007) and neurobehavioral tasks (Ceballos et al., 2005; Ceballos et al., 2006; Gilbertson et al., 2011). Notably, to reduce the effects of nicotine withdrawal, many of these studies included only active doses.

The current study replicated and extended previous work on attentional and working memory processes in recently abstinent substance abusers using a double-blind, placebo-controlled design. Given existing work with nicotine, we anticipated placebo would result in poorer performance than active doses and that active doses would be accompanied by dose-dependent enhancement. Furthermore, we posited that the alcoholic group would demonstrate greater benefit than the control or stimulant abusing groups on the tasks under study, while recognizing that the mechanism underlying this effect is unclear. The inclusion of stimulant abusing groups provides essential information on the potential effects of substance abuse history on nicotine effects in substance abusing populations.

Finally, recent work has identified smoking history as a significant negative predictor of recovery of biomarkers associated with neural function (Durazzo et al., 2006a) and medial temporal lobe volume (Gazdzinski et al., 2008), as well as a significant source of variation in neurocognitive performance among abstinent alcoholics (Durazzo et al., 2006b). Thus, while acute nicotine use may benefit neurocognitive function, chronic smoking appears to have negative effects. Simultaneous consideration of these opposing effects has not been previously reported. Based on current literature, we predicted that intensity of chronic smoking would be associated with poorer performance. We empirically questioned whether the effect would be equally experienced across smoking subgroups.

2. Methods

2.1 Participants (Subjects, Ss)

This study included recently abstinent substance abusers (SAs; N = 86) recruited from inpatient treatment facilities near Gainesville, FL and Lexington, KY. Participants were subgrouped on the basis of substance use diagnoses: alcohol dependency without co-morbid stimulant dependence (ALCS; n=24, 12 women), alcohol dependency with co-morbid stimulant dependence (ALC/STIMS; n=44, 13 women) and stimulant dependency (STIMS; n=18, 9 women). 96% of STIMS reported cocaine as their drug of choice; the remaining 4% reported amphetamines. SAs were 15–86 days abstinent from their drug of choice (M=40.3, SD=16.64). 89% of SAs were current smokers. Therefore, it was impracticable to recruit non-smoking SAs. Community-dwelling smokers (smoking CNTLS; n=17, 9 women) and non-smokers (non-smoking CNTLS; n=22, 14 women) were also recruited. Substance use disorders, excepting nicotine for smokers, were exclusionary for CNTLS. Ss ranged in age from 22–58 with 10–18 years of education. The study was IRB approved. Ss provided informed consent prior to participation.

2.2 Screening

Screening consisted of pencil/paper questionnaires assessing demographics, affective state (Beck et al., 1996; Spielberger, 1983) and alcohol/drug use histories. Exclusionary psychiatric diagnoses were assessed with the computerized Diagnostic Interview Schedule IV (cDIS-IV; Robins, 1995; APA, 1994). Individuals with diagnoses not consistent with assigned group were excluded, as were those with medical histories that might confound cognitive measures or contraindicate patch administration. A breath-based carbon monoxide (CO) measure (Vitalograph Inc., Lenexa, KS) provided at screening served as the comparison for verification of short term abstinence at laboratory testing.

2.3 Laboratory Testing

As in previous work, Ss were advised to not smoke overnight prior to the study (Hughes, 1991; Pritchard and Robinson, 1998; Heishman et al., 2010). Abstinence was confirmed by CO testing (see Cinciripini et al., 2006; Nixon et al., 2007; SRNT SBV, 2002). Positive urine and breath tests for recent illicit drug (OnTrak TestCup 5®, Varian, Inc., Palo Alto, CA) and ethanol (Intoxylizer® 400PA, CMI, Inc., Owensboro, KY) use were exclusionary. Pregnant/breastfeeding women were also excluded.

SAs and smoking CNTLS were assigned placebo (0 mg), low (7 mg) or high (14/21 mg; female/male, respectively) dose transdermal nicotine patches (Nicoderm CQ, GlaxoSmithKline, London, England). Because the 21 mg dose has resulted in adverse effects in women in our lab and others (e.g., Evans et al., 2006), 14 mg patches constituted the high dose in women. Assignments were made according to a 4 (ALC; ALC/STIM; STIM; smoking CNTL) X 3 (Placebo/Low/High) randomized, double-blind experimental design. To minimize adverse events, non-smoking CNTLS received only placebo or 7 mg patch. Staff unassociated with testing applied the patch. Testing was delayed ~2 hours to allow nicotine levels to stabilize (Benowitz et al., 1993). After testing, Ss were reimbursed, debriefed and informed of their dose. Ss receiving active doses were advised to avoid smoking for several hours.

2.4 Rapid Visual Information Processing (RVIP) Task

The RVIP (Wesnes and Warburton, 1983) is a vigilance and working memory test widely used in acute nicotine studies (Edwards et al., 1985; Mancuso et al., 1999; Ceballos et al., 2006). Ss pressed a button when three consecutive odd or even digits were presented in a series of digits (presented at 1.6/sec). Accuracy (ACC) and reaction time (RT) were DVs;

neither was significantly skewed or kurtosed (Tabachnick and Fidell, 2001). No corrective transformations were applied.

3. Results

SAS 9.1 (SAS Institute, Inc., Cary, NC) was used for all data analysis.

Demographic/Substance Use

Significant differences were detected in demographic variables between groups ($F_{8,236}=6.01$, $p<.0001$). Follow-up univariate analyses adjusting for unequal cells were followed by paired comparisons, which showed differences only in years of education. Non-smoking CNTLS ($M=15.61$, $SD=2.18$) had more years of education than ALCS ($M=13.38$, $SD=1.74$; $t_{45}=4.09$, $p<.0001$), ALC/STIMS ($M=12.80$, $SD=1.66$; $t_{65}=5.80$, $p<.0001$) and STIMS ($M=12.72$, $SD=1.96$; $t_{39}=4.91$, $p<.0001$).

Measures of anxiety and depression also differed by group ($F_{8,198}=5.03$, $p<.0001$). Non-smoking CNTLS ($M=41.29$, $SD=4.07$) endorsed less anxiety than ALCS ($M=55.09$, $SD=13.07$; $t_{45}=4.25$, $p<.0001$) and ALC/STIMS ($M=52.07$, $SD=10.37$; $t_{65}=3.11$, $p=.003$); ALCS also had significantly greater anxiety than smoking CNTLS ($M=44.21$, $SD=7.53$; $t_{40}=3.42$, $p=.0009$). ALCS ($M=14.53$, $SD=9.00$) endorsed greater depressive symptomatology than any other group ($M_{\text{overall}}=6.43$, $SD=5.85$; $t's>2.80$, $p's<.006$) and ALC/STIMS ($M=9.23$, $SD=6.13$) endorsed greater depression than non-smoking CNTLS ($M=2.50$, $SD=3.14$; $t_{65}=3.31$, $p=.001$). These measures did not reflect significant distress.

Differences were detected in nicotine-related variables ($F_{21,259}=1.70$, $p=.03$; non-smoking CNTLS excluded). Test-day CO measures were greater in smoking CNTLS ($M=11.50$ ppm, $SD=5.62$) than in STIMS ($M=8.39$, $SD=4.02$; $t_{34}=2.05$, $p=.04$) or ALC/STIMS ($M=8.07$, $SD=3.45$; $t_{60}=2.66$, $p=.009$), although all subjects met test day criteria for smoking abstinence. Assessments of withdrawal symptomatology did not differ by nicotine dose ($F_{2,100}=1.73$, $p=.18$).

As expected, groups differed significantly in the quantity frequency index (QFI, average ounces of absolute ethanol consumed per day; $F_{4,129}=31.21$, $p<.0001$) such that ALC ($M=11.86$, $SD=6.10$) > ALC/STIM ($M=7.95$, $SD=5.18$) > STIM ($M=1.37$, $SD=1.54$) = smoking CNTL ($M=0.55$, $SD=0.46$) = non-smoking CNTL ($M=0.24$, $SD=0.29$) ($t's>3.70$, $p's<.0003$).

3.2 Correlational Analyses

Demographic, affective, and substance use variables failed to correlated with DVs ($r's<.18$, ns). Correlation matrices (Pearson's r) revealed that pack-years (a composite measure of smoking intensity and chronicity; Prigot, 1987) correlated with poorer ACC and RT ($r=-.17$, $p=.08$ and $r=.19$, $p=.05$, respectively). When analyzed by group, the correlation was significant only for ALCS (RVIP: $r=-.58$, $p=.003$ and $r=.53$, $p=.007$; for all other groups, $r<.23$, ns). As expected, age and pack-years were highly correlated ($r=.59$, $p<.0001$). Age did not correlate with performance measures ($r<.17$, ns).

3.3 Behavioral Performance

3.3.1 RVIP—Although not of primary interest, non-smoking CNTLS demonstrated expected nicotine-related benefit on ACC, although not statistically significant ($F_{1,20}=3.83$, $p=.06$). There were no differences in performance between women receiving 14 mg patches and men receiving 21 mg ($p's>.40$). 4 (Group: ALC, ALC/STIM, STIM, smoking CNTL) X 3 (Nicotine dose: Placebo, Low, High) analysis conducted using general linear models

revealed a significant group X patch interaction for RT ($F_{6,88}=2.24$, $p=.05$) and a main effect of group for ACC ($F_{3,91}=3.07$, $p=.03$). Follow-up univariate analyses adjusting for unequal cells were followed by paired comparisons. A significant effect of nicotine dose on RT in smoking CNTLS ($F_{2,13}=21.3$, $p<.0001$) was found; the high dose was associated with faster RT than the low ($t_{10}=5.35$, $p=.0001$) or placebo ($t_8=6.10$, $p<.0001$) doses. Unexpectedly, smoking CNTLS ($M=33.88$, $SD=18.66$) had poorer ACC than ALCS ($M=44.00$, $SD=22.74$; $t_{40}=-2.23$, $p=.03$) or STIMS ($M=39.18$, $SD=19.64$; $t_{34}=-2.10$, $p=.04$).

Given the previously noted correlation between pack-years and performance measures in ALCS, univariate analyses examined potential nicotine dose effects (controlling for pack-years) in this group. This analysis revealed trend-level nicotine effects on ACC in ALCS ($F_{2,20}=3.20$, $p=.06$; Fig. 1). Planned comparisons revealed the high dose of nicotine was associated with better ACC than the low dose ($t_{19}=2.49$; $p=.02$) and placebo (although nonsignificant; $t_{17}=1.67$, $p=.11$) which did not differ ($p>.40$).

To clarify the role of pack-years, stepwise linear regression was conducted in ALCS. Results indicated that for this group, pack-years predicted poorer performance for both RT ($F_{1,25}=8.69$, $p=.007$; $r=.53$, $R^2=.28$) and ACC ($F_{1,22}=11.40$, $p=.003$; $r=-.58$, $R^2=.34$) (Fig. 2). Because pack-years and performance variables were not correlated in other groups, no additional analysis was done.

4. Discussion

4.1 Overview

These results support previous work indicating a compensatory effect of nicotine on neurocognition in treatment-seeking ALCS. We found that alcoholics were sensitive to nicotine's performance-enhancing effects on vigilance and working memory performance when smoking history is considered (Ceballos et al., 2006; Nixon et al., 2007). This enhancement may represent a dose threshold effect; i.e., the high dose was associated with greater accuracy in ALCS than the low dose or placebo. In smoking CNTLS, the benefit of nicotine was restricted to RT. Their poor accuracy at the high dose (mean=20.33, $SD=5.51$) suggests subjects produced anticipatory and inaccurate responses. Finally, no effect of nicotine on accuracy or reaction time was detected for the stimulant abusing subgroups. Although more research is required, this may be due to a modulatory effect of a history of stimulant dependence on acute nicotine effects.

Analysis of the relationship between pack-years and performance revealed that smoking history significantly predicted poorer performance in ALCS. These findings are preliminary, yet complementary to recent work suggesting chronic smoking is a significant source of variation in neurocognition and its recovery during early abstinence in alcoholics (Durazzo et al., 2006a; Durazzo et al., 2006b; Durazzo and Meyerhoff, 2007; Gazdzinski et al., 2008; Meyerhoff et al., 2006; Mon et al., 2009).

4.1 Limitations

Although intriguing, these findings are limited by several factors. First, recruitment difficulties resulted in a small sample of smoking CNTLS administered the high dose ($n=3$). Therefore, conclusions regarding nicotine effects in this group should be studied further. Secondly, although potential interactions of sex and nicotine dose are of interest, there were insufficient numbers of women across groups to conduct meaningful analysis.

4.2 Conclusions

In summary, this study of the effects of acute nicotine administration on vigilance and working memory task performance provides additional evidence for the sensitivity of detoxified, currently-smoking alcoholics to the cognitive enhancing effects of nicotine. Furthermore, the results indicating that alcoholics' smoking history significantly predicts task performance are provocative and justify further programmatic study. Although the study has limits, the findings justify additional research regarding the relationship between alcoholics' smoking history, current nicotine use, and neurocognitive capacity in early recovery.

Abbreviations

STIM	Stimulant-dependent individual
ALC	Alcoholic without co-morbid stimulant dependence
ALC/STIM	Alcoholic with co-morbid stimulant dependence
CNTL	Community-dwelling control
RVIP	Rapid Visual Information Processing Task
DV	Dependent variable
SA	Substance abuser

References

- American Psychiatric Association Task Force on DSM-IV. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: American Psychiatric Association; 1994.
- Beck, AT.; Steer, RA.; Brown, GK. Beck Depression Inventory. 2. San Antonio: The Psychological Corporation; 1996.
- Bell SL, Taylor RC, Singleton EG, Henningfield JE, Heishman SJ. Smoking after nicotine deprivation enhances cognitive performance and decreases tobacco craving in drug abusers. *Nicotine Tob Res.* 1999; 1(1):45–52. [PubMed: 11072387]
- Benowitz NL. Nicotine replacement therapy. What has been accomplished--can we do better? *Drugs.* 1993; 45(2):157–170. [PubMed: 7681369]
- Ceballos NA, Tivis R, Lawton-Craddock A, Nixon SJ. Visual-spatial attention in alcoholics and illicit stimulant abusers: effects of nicotine replacement. *Prog Neuropsychopharmacol Biol Psychiatry.* 2005; 29(1):97–107. [PubMed: 15610951]
- Ceballos NA, Tivis R, Lawton-Craddock A, Nixon SJ. Nicotine and cognitive efficiency in alcoholics and illicit stimulant abusers: implications of smoking cessation for substance users in treatment. *Subst Use Misuse.* 2006; 41(3):265–281. [PubMed: 16467005]
- Cinciripini PM, Robinson JD, Carter BL, Lam C, Wu X, de Moor CA, et al. The effects of smoking deprivation and nicotine administration on emotional reactivity. *Nicotine Tob Res.* 2006; 8(3):379–392. [PubMed: 16801296]
- Durazzo TC, Gazdzinski S, Rothlind JC, Banys P, Meyerhoff DJ. Brain metabolite concentrations and neurocognition during short-term recovery from alcohol dependence: Preliminary evidence of the effects of concurrent chronic cigarette smoking. *Alcohol Clin Exp Res.* 2006; 30(3):539–551. [PubMed: 16499496]
- Durazzo TC, Meyerhoff DJ. Neurobiological and neurocognitive effects of chronic cigarette smoking and alcoholism. *Front Biosci.* 2007; 12:4079–4100. [PubMed: 17485360]
- Durazzo TC, Rothlind JC, Gazdzinski S, Banys P, Meyerhoff DJ. A comparison of neurocognitive function in nonsmoking and chronically smoking short-term abstinent alcoholics. *Alcohol.* 2006; 39(1):1–11. [PubMed: 16938624]

- Edwards JA, Wesnes K, Warburton DM, Gale A. Evidence of more rapid stimulus evaluation following cigarette smoking. *Addict Behav.* 1985; 10(2):113–126. [PubMed: 4013861]
- Evans SE, Blank M, Sams C, Weaver MF, Eissenberg T. Transdermal nicotine-induced tobacco abstinence symptom suppression: nicotine dose and smokers' gender. *Exp Clin Psychopharmacol.* 2006; 14(2):121–135. [PubMed: 16756416]
- Gazdzinski S, Durazzo TC, Yeh PH, Hardin D, Banys P, Meyerhoff DJ. Chronic cigarette smoking modulates injury and short-term recovery of the medial temporal lobe in alcoholics. *Psychiatry Res.* 2008; 162(2):133–145. [PubMed: 18178068]
- Gilbertson RJ, Boissoneault JB, Prather R, Nixon SJ. Nicotine Effects on Immediate and Delayed Verbal Memory After Substance Use Detoxification. *J Clin Exp Neuropsychol.* 2011
- Heishman SJ, Kleykamp BA, Singleton EG. Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacology (Berl).* 2010; 210(4):453–469. [PubMed: 20414766]
- Hughes JR. Distinguishing withdrawal relief and direct effects of smoking. *Psychopharmacology (Berl).* 1991; 104(3):409–410. [PubMed: 1924648]
- Kumari V, Gray JA, ffytche DH, Mitterschiffthaler MT, Das M, Zachariah E, et al. Cognitive effects of nicotine in humans: an fMRI study. *Neuroimage.* 2003; 19(3):1002–1013. [PubMed: 12880828]
- Lawrence NS, Ross TJ, Stein EA. Cognitive mechanisms of nicotine on visual attention. *Neuron.* 2002; 36(3):539–548. [PubMed: 12408855]
- Levin ED, McClernon FJ, Rezvani AH. Nicotinic effects on cognitive function: behavioral characterization, pharmacological specification, and anatomic localization. *Psychopharmacology (Berl).* 2006; 184(3–4):523–539. [PubMed: 16220335]
- Littleton J, Barron S, Prendergast M, Nixon SJ. Smoking kills (alcoholics)! shouldn't we do something about it? *Alcohol Alcohol.* 2007; 42(3):167–173. [PubMed: 17526626]
- Mancuso G, Andres P, Ansseau M, Tirelli E. Effects of nicotine administered via a transdermal delivery system on vigilance: a repeated measure study. *Psychopharmacology (Berl).* 1999; 142(1):18–23. [PubMed: 10102778]
- Meyerhoff DJ, Tizabi Y, Staley JK, Durazzo TC, Glass JM, Nixon SJ. Smoking comorbidity in alcoholism: neurobiological and neurocognitive consequences. *Alcohol Clin Exp Res.* 2006; 30(2):253–264. [PubMed: 16441274]
- Mon A, Durazzo TC, Gazdzinski S, Meyerhoff DJ. The impact of chronic cigarette smoking on recovery from cortical gray matter perfusion deficits in alcohol dependence: longitudinal arterial spin labeling MRI. *Alcohol Clin Exp Res.* 2009; 33(8):1314–1321. [PubMed: 19413652]
- Nixon SJ, Lawton-Craddock A, Tivis RD, Ceballos NA. Nicotine's effects on attentional efficiency in alcoholics. *Alcohol Clin Exp Res.* 2007; 31(12):2083–2091. [PubMed: 17949466]
- Oscar-Berman M, Marinkovic K. Alcohol: effects on neurobehavioral functions and the brain. *Neuropsychol Rev.* 2007; 17(3):239–257. [PubMed: 17874302]
- Pfefferbaum A, Adalsteinsson E, Sullivan EV. Dismorphology and microstructural degradation of the corpus callosum: Interaction of age and alcoholism. *Neurobiol Aging.* 2006; 27(7):994–1009. [PubMed: 15964101]
- Pfefferbaum A, Sullivan EV, Mathalon DH, Shear PK, Rosenbloom MJ, Lim KO. Longitudinal changes in magnetic resonance imaging brain volumes in abstinent and relapsed alcoholics. *Alcohol Clin Exp Res.* 1995; 19(5):1177–1191. [PubMed: 8561288]
- Prignot J. Quantification and chemical markers of tobacco-exposure. *Eur J Respir Dis.* 1987; 70(1):1–7. [PubMed: 3545884]
- Pritchard, WS.; Robinson, JH. Effects of nicotine on human performance. In: Snel, J.; Lorist, MM., editors. *Nicotine, caffeine and social drinking: behaviour and brain function.* Amsterdam: Harwood Academic Publishers; 1998. p. 21–81.
- Rezvani AH, Levin ED. Cognitive effects of nicotine. *Biol Psychiatry.* 2001; 49(3):258–267. [PubMed: 11230877]
- Robins, LN.; Cottler, L.; Bucholz, KK.; Compton, W. *The Diagnostic Interview Schedule, Version IV.* St. Louis: Washington University; 1995.
- Spielberger, CD. *Manual for State-Trait Anxiety Inventory.* Palo Alto, CA: Consulting Psychologists Press; 1983.

Society for Research on Nicotine and Tobacco Subcommittee on Biochemical Verification (SRNT SBV). Biochemical verification of tobacco use and cessation. *Nicotine Tob Res.* 2002; 4(2):149–159. [PubMed: 12028847]

Tabachnick, BG.; Fidell, LS. *Using Multivariate Statistics*. 2. Glenview, Illinois: HarperCollins; 1989.

Wesnes K, Warburton DM. Effects of smoking on rapid information processing performance. *Neuropsychobiology.* 1983; 9(4):223–229. [PubMed: 6646394]

Highlights

- This was a placebo controlled study of regular smokers.
- Acute nicotine effects on cognition were studied.
- Performance was measured on attention/working memory tasks.
- Nicotine improved task performance in non-stimulant dependent alcoholics.
- Intensity of chronic smoking predicted poorer performance in alcoholics.

Figure 1.

In ALCS, the high dose of nicotine was associated with better accuracy compared to the low dose ($p=.02$). A similar trend was noted for the high dose vs. placebo comparison ($p=.11$).

Figure 2.

Increasing pack-years significantly predicted less accurate performance and longer RT among ALCS in the RVIP task ($p=.007$; $r=-.53$, $R^2=.28$; $p=.003$; $r=.58$, $R^2=.34$; accuracy & RT, respectively).